

Millie Jones Case Study Answer Key: Dyslipidemia and CKD

Objectives

1. Student will associate normal urine albumin-to-creatinine ratios (UACR) with slower progression of chronic kidney disease (CKD).
2. Student will identify diet strategies that may reduce lipids in CKD.
3. Student will identify total 25(OH)D as the marker for vitamin D status in CKD.
4. Student will identify hemoglobin as marker of anemia and transferrin saturation and ferritin as iron indices used to assess iron status in CKD.

Background

Mrs. Millie Jones is a 79-year-old white woman with 30 year history of type 2 diabetes, hypertension, hyperlipidemia, and CKD. She feels a lot better since the statin was discontinued for muscle aches and pain (myopathy) last September and since then increased her daily walks to 45 minutes a day. No tobacco or alcohol use. Was treated for a urinary tract infection last August.

Physical exam: thin woman, upper dentures. No obvious nutrient deficiencies.

MNT Referral Data

Referred for dyslipidemia. Myopathy with statin, starting ezetimibe 10 milligrams.

Labs: Hemoglobin A1C 6.7, UACR 18.7, creatinine 2.1, eGFR 26, K 4.0, HCO₃ 26.4, BUN 32, Ca 9.2, Phos 4.0, Hgb 12.1, LDL 185, HDL 39, TG 176, iPTH 165, Vit D 72, Alb 4.0

Medications: Losartan 20 milligrams (mg) daily, furosemide 40 mg daily, baby aspirin, ferrous sulfate 325 mg twice a day, levothyroxine 25 micrograms daily, ergocalciferol 50,000 I.U. weekly, calcium carbonate 1000 mg daily.

Recall

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| <ul style="list-style-type: none"> ½ cup oatmeal 4 oz. 2% milk 1 boiled egg 1 slice white toast ½ tbsp. butter 2 cups black coffee | <ul style="list-style-type: none"> 1 slice deli roast beef (1 ½ oz.) 1 slice American cheese 2 slices wheat bread Lettuce/tomato/onion/mayo Small handful of potato chips 1 can diet lemon lime soda pop | <ul style="list-style-type: none"> 1 baked chicken leg, no skin ½ cup green beans (frozen kind) 1 small baked potato/ 1 tbsp. butter ½ cup canned light peaches 1 cup hot tea with lemon |
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Review of Pertinent Measures

| Measure | Reference Range | 2/4/12 | 10/10/11 | 9/29/11 | 8/28/11 | 6/16/11 |
|------------------|-----------------|--------|----------|---------|---------|---------|
| UACR | < 30 | 18.7 | --- | --- | --- | 14.2 |
| Glucose | 70-100 | 122 H | 201 H | 142 H | 135 H | 128 H |
| BUN | 7-20 | 32 H | 30 H | 43 H | 77 H | 48 H |
| Creatinine | 0.8-1.3 | 2.1 H | 2.3 H | 2.6 H | 3.5 H | 2.4 H |
| eGFR | > 60 | 26 L | 22 L | 19 L | 14 L | 21 L |
| Sodium | 135-145 | 139 | 140 | 138 | 142 | 142 |
| Potassium | 3.5-5.0 | 4.0 | 3.8 | 4.2 | 4.8 | 5.0 |
| Chloride | 101-111 | 102 | 104 | 104 | 110 | 106 |
| HCO ₃ | 21-32 | 26.4 | 24.9 | 22.4 | 17.8 L | 24.3 |
| Calcium | 8.5-10.2 | 9.2 | 8.8 | 9.0 | 8.6 | 9.0 |
| Phosphorus | 2.7-4.6 | 4.0 | 4.0 | 4.2 | 4.8 H | 4.4 |
| Albumin | 3.4-5.0 | 4.0 | 3.8 | 4.1 | 3.7 | 3.6 |
| Cholesterol | < 200 | 271 H | --- | --- | --- | 154 |
| LDL | < 100 | 185 H | --- | --- | --- | 72.8 |
| HDL | > 35 | 39 | --- | --- | --- | 36 |
| Triglycerides | < 150 | 176 H | --- | --- | --- | 231 H |
| 25(OH) D | 20 or more | 72 | --- | 30 | --- | < 4 L |
| iPTH | 65 or less | 165 H | | 232 H | --- | --- |
| Hemoglobin A1C | < 7 % | 6.7 | 5.9 | 6.0 | 6.6 | 5.9 |

Questions

For additional information, see noted slides from **Chronic Kidney Disease 101: Nutrition Intervention**, available at <http://nkdep.nih.gov/resources/ckd-101-nutrition-508.ppt>.

1. **What is your assessment of her kidney function and risk of progression to kidney failure?**

Use NKDEP's *How well are your kidneys working? Explaining your kidney test results* (<http://nkdep.nih.gov/resources/explaining-kidney-test-results-508.pdf>) to show eGFR and UACR results and refer to NKDEP's *Quick Reference on UACR and GFR* (<http://www.nkdep.nih.gov/resources/quick-reference-uacr-gfr-508.pdf>).

Answer: Her eGFR declined acutely and is back to baseline. Overall, kidney function is reduced, and appears to be stabilized after a urinary tract infection was treated and resolved. Her risk of progression is lower as evidenced by a normal UACR level. Her eGFR is significantly reduced and UACR is normal; this is atypical for kidney disease due to diabetes and may be due to another etiology.

2. She was referred for dyslipidemia. What are the key findings in the lipid results?

Answer: Total cholesterol, LDL cholesterol and triglycerides are elevated. Total and LDL cholesterol increased since the statin was discontinued.

For additional information, see slides 22, 43, 47, 48.

3. List at least 3 dietary changes she could consider to help lower lipids.

Possible Answers:

- Replace butter with soft margarine made without hydrogenated fats.
- Eat a piece of fruit rich in soluble fiber in place of potato chips for lunch.
- Use skim or 1% milk in place of 2% milk.
- Reduce cheese intake.
- Substitute home cooked turkey breast (not enhanced) instead of deli roast beef.

For additional information, see slides 43-46.

4. Briefly explain the role of the kidneys in vitamin D status.

Answer: The kidneys are the site of the final hydroxylation of 25(OH) vitamin D to its active form 1,25 (OH)₂ vitamin D by the 1-alpha-hydroxylase enzyme.

For additional information, see slides 5, 60, 63 (notes).

5. Explain the role of parathyroid hormone (PTH) in chronic kidney disease.

Answer: The parathyroid maintains serum calcium and has an indirect role in maintaining serum phosphorus. Low serum calcium stimulates the parathyroid gland to secrete additional parathyroid hormone. PTH increases calcium resorption from bone and increases intestinal calcium absorption by stimulating the enzyme (1-alpha-hydroxylase) in the kidney responsible for the final activation of vitamin D. PTH also increases urinary excretion of phosphorus. In CKD, serum phosphorus levels may remain in the normal range as a result of higher PTH levels. Serum phosphorus levels may increase as the eGFR decreases. Vitamin D and PTH may be inversely and supplementation with vitamin D may lower PTH levels.

For additional information, see slides 5 (notes), 59-61, 63, 83.

6. What are the key findings in her iPTH, vitamin D, and serum phosphorus levels?

Answer: Intact PTH is elevated but decreased as vitamin D level increased, and serum phosphorus is still within normal range.

For additional information, see slides 5, 59-61, 63, 83.

7. Document the visit.

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| A. | 79 yo woman, type 2 diabetes X 30 years. Ht: 60; Wt: 125# (stable). Pertinent labs: LDL 185. Stopped statin due to myopathy, starting ezetimibe. |
| D. | Excessive saturated fat intake related to lack of knowledge of dietary management of lipids as evidenced by diet recall indicating use of foods rich in saturated fat. |
| I. | Nutrition education, priority modification and relationship to health and disease: Lower saturated fat intake and relationship to controlling lipids. |
| M&E. | Short term: Patient stated she will use soft margarine in place of butter. Long term: Recheck diet recall in the future. Monitor LDL for improvement. |

Follow Up with Mrs. Jones

Mrs. Jones returns for follow-up as planned. She found a soft margarine without hydrogenated oil but uses very little on her foods now. She is now concerned about her blood count results. Last year, her physician told her to take ferrous sulfate 325 milligrams twice a day. The iron supplement causes diarrhea and she needs to stay home when she takes it. She only takes it once a day now. She wants to know if she still needs to take it.

Review of Pertinent Measures

| Measure | Reference Range | 4/27/12 | 2/4/12 | 10/10/11 | 9/29/11 |
|------------------------------------|-----------------|---------|--------|----------|---------|
| HGB | 12-16 | 12.6 | 12.1 | 11.3 L | 11.2 L |
| HCT | 37-47 | 38.1 | 38.5 | 32.1 L | 31.4 L |
| Total iron binding capacity (TIBC) | 250-450 | 364 | --- | --- | 300 |
| Serum iron | 40-160 | 66 | --- | --- | 60 |
| Transferrin saturation % (TSAT) | 15-50 % | 18 | --- | --- | 17 |
| Serum ferritin | 20-288 | 68 | --- | --- | 127 |

Additional Questions

8. Briefly explain the role of the kidneys in anemia. What does this mean for someone with chronic kidney disease (CKD)?

Answer: The kidneys are the main source of erythropoietin, the hormone needed for red blood cell synthesis within bone marrow. People with chronic kidney disease are at risk for anemia as kidney function declines.

For additional information, see slides 5, 59-61, 63, 83.

9. What is the key finding in her blood count results?

Answer: Hemoglobin level is increasing with the use of oral iron.

For additional information, see slides 70-73, 82.

10. Briefly describe the major difference between transferrin saturation and ferritin.

Answer: The iron bound to transferrin reflects the biologically available iron. Ferritin is a reflection of total body iron stores.

For additional information, see slide 70.

11. Which of the following strategies is the best way to address her concern about supplemental iron?

Answer: b. You will discuss her concerns with her physician and get back to her. He may recommend a different iron formulation.

For additional information, see slides 10, 70-73.

Educational Material

National Kidney Disease Education Program. *How well are your kidneys working? Explaining your kidney test results*. Revised February 2012. NIH Publication No.12–6220. National Kidney Disease Education Program website. <http://www.nkdep.nih.gov/resources/explaining-kidney-test-results-508.pdf>

National Kidney Disease Education Program. *Urine Albumin-to-Creatinine Ratio (UACR) in evaluating patients with diabetes for kidney disease*. Washington, D.C.: U.S. Government Printing Office; 2010. NIH Publication No.10–6286. National Kidney Disease Education Program website. <http://www.nkdep.nih.gov/resources/quick-reference-uacr-qfr-508.pdf>

Additional Reading

Beto JA, Bansal VK. Nutrition interventions to address cardiovascular outcomes in chronic kidney disease. *Advances in Chronic Kidney Disease*. 2004;11(4):391-397.

Martin KJ, González EA. Metabolic bone disease in chronic kidney disease. *Journal of the American Society of Nephrology*. 2007;18(3):875-885.

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Peacock, M. Calcium metabolism in health and disease. *Clinical Journal of the American Society of Nephrology*. 2010; 5:S23-S30.

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Felsenfeld AJ, Rodriguez M, Aguilera-Tejero E. Dynamics of parathyroid hormone secretion in health and secondary hyperparathyroidism. *Clinical Journal of the American Society of Nephrology*. 2007;2(6):1283–1305. <http://cjasn.asnjournals.org/content/2/6/1283.full.pdf+html>

Kovesdy CP, Estrada W, Ahmadzadeh S, Kalantar-Zadeh K. Association of markers of iron stores with outcomes in patients with nondialysis-dependent chronic kidney disease. *Clinical Journal of the American Society of Nephrology*. 2009; 4(2): 435-441.

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Besarab A, Coyne DW. Iron supplementation to treat anemia in patients with chronic kidney disease. *Nature Reviews Nephrology*. 2010; 6: 699-710.

Kovesdy CP. Iron and clinical outcomes in dialysis and non-dialysis dependent chronic kidney disease patients. *Advances in Chronic Kidney Disease*. 2009;16(2):109-116.



For more information, visit www.nkdep.nih.gov/nutrition or call 1-866-4 KIDNEY (1-866-454-3639).

The National Kidney Disease Education Program (NKDEP) works to improve the understanding, detection, and management of kidney disease. NKDEP is a program of the National Institutes of Health.

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